

AMENDMENTS TO THE CLAIMS

The claims in this listing will replace all prior versions, and listings, of claims in the application.

Listing of Claims:

1. (Original): A pharmaceutical composition for preventing and treating dementia, which contains minocycline as an active ingredient.
2. (Original): The pharmaceutical composition of Claim 1, which inhibits brain cell toxicity.
3. (Original): The pharmaceutical composition of Claim 2, which inhibits the brain cell toxicity of amyloid beta-protein.
4. (Original): The pharmaceutical composition of Claim 2, which inhibits the brain cell toxicity of C-terminal protein.
5. (Original): The pharmaceutical composition of Claim 1, which inhibits the impairment of learning and memory and cognitive function.
6. (Original): The pharmaceutical composition of Claim 5, which inhibits the impairment of learning and memory and cognitive function induced by amyloid beta-protein.

7. (Original): The pharmaceutical composition of Claim 5, which inhibits the impairment of learning and memory and cognitive function induced by C-terminal amyloid precursor protein.

8. (Currently amended): The pharmaceutical composition of ~~any one of Claims 1 to 7~~ Claim 1, wherein the dementia is Alzheimer's disease.

9. (Original): A pharmaceutical composition for preventing and treating the impairment of learning and memory and cognitive function which contains minocycline as an active ingredient.

10. (Original): The pharmaceutical composition of Claim 9, which inhibits brain cell toxicity.

11. (Original): The pharmaceutical composition of Claim 10, which inhibits the brain cell toxicity of amyloid beta-protein.

12. (Original): The pharmaceutical composition of Claim 10, which inhibits the brain cell toxicity of C-terminal protein.

13. (Original): The pharmaceutical composition of Claim 9, which inhibits the impairment of learning and memory and cognitive function induced by amyloid beta-protein.

14. (Original): The pharmaceutical composition of Claim 9, which inhibits the impairment of learning and memory and cognitive function induced by C-terminal of amyloid precursor protein.